L18 ANSWER 4 OF 24 MEDLINE
ACCESSION NUMBER: 1999397656 MEDLINE
DOCUMENT NUMBER: 99397656 PubMed ID: 10471055
TITLE: Analysis of potential markers for detection of submicroscopic lymph node **metastases** in breast cancer.
AUTHOR: Merrie A E; Yun K; Gunn J; Phillips L V; McCall J L
CORPORATE SOURCE: Department of Surgery, Dunedin School of Medicine, University of Otago, New Zealand.
SOURCE: BRITISH JOURNAL OF CANCER, **(1999 Aug)** 80 (12) 2019-24.
Journal code: 0370635. ISSN: 0007-0920.
PUB. COUNTRY: SCOTLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199909
ENTRY DATE: Entered STN: 19991005
Last Updated on STN: 20000303
Entered Medline: 19990921

AB We have developed sensitive assays for cytokeratin (K) 8, 16, 19, stromelysin 3 (ST3), MUC1 and **maspin** mRNAs using reverse transcription polymerase chain reaction (RT-PCR) and used these to assess lymph node status in patients undergoing surgery for breast cancer. In addition the RT-PCR assays were tested against lymph nodes from non-cancer patients to determine their specificity. Despite high sensitivity RT-PCR assays for K8, K16, K19, ST3 and **maspin** were not found to be useful as markers of submicroscopic disease as transcripts of these genes were detected in the great majority of control lymph nodes tested. Expression of MUC1 was also not found to be useful as it was both insensitive and non-specific. The importance of assessing potential markers against an adequately sized control population is demonstrated, as failure to do so can lead to erroneous conclusions.

L18 ANSWER 7 OF 24 MEDLINE
ACCESSION NUMBER: 1998031853 MEDLINE
DOCUMENT NUMBER: 98031853 PubMed ID: 9366525
TITLE: Evidence of a dominant transcriptional pathway which regulates an undifferentiated and complete **metastatic** phenotype.
AUTHOR: Barsky S H; Sternlicht M D; Safarians S; Nguyen M; Chin K; Stewart S D; Hiti A L; Gray J W
CORPORATE SOURCE: Department of Pathology, University of California Los Angeles School of Medicine 90024, USA.
CONTRACT NUMBER: CA01351 (NCI)
CA40225 (NCI)
CA56735 (NCI)
+
SOURCE: ONCOGENE, **(1997 Oct 23)** 15 (17) 2077-91.
Journal code: 8711562. ISSN: 0950-9232.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971120

AB The highly metastatic amelanotic C8161 human melanoma line was found to exhibit complete dominance of its undifferentiated and metastatic phenotype in multiple somatic cell hybridization studies designed to bypass the presence of potential tumor suppressor genes. In a three armed approach involving somatic cell fusions of C8161 with recipient lines of greater differentiation, different lineage, and different tumorigenicity status, the metastatic and undifferentiated phenotype of C8161 was promiscuously dominant. In somatic cell hybrids produced between the C8161 and a group of non-metastatic human melanoma lines which exhibited melanocyte differentiation markers including S100, HMB-45, NKI/C3, and melanin, the fusions were uniformly metastatic and undifferentiated. In somatic cell hybrids of C8161 and MCF-7 the fusions exhibited an estrogen independent and unresponsive, estrogen receptor (ER) negative, and highly metastatic phenotype. In fusions between C8161 and HMS-1, an immortalized 'benign' human myoepithelial line which produced an abundant extracellular matrix (ECM) and high levels of protease and angiogenic inhibitors including **maspin**, tissue inhibitor of metalloproteinase-1 (TIMP-1), alpha1-antitrypsin (alpha1-AT), protease nexin II (PN-II), thrombospondin-1 and soluble basic fibroblast growth factor (bFGF) receptors, the hybrids showed complete absence of matrix, absent **maspin** expression, markedly decreased protease inhibitor and angiogenic inhibitor production, high levels of proteases and angiogenic factors, and a highly metastatic phenotype. In our somatic cell fusions, the human-human hybrids represented true and complete fusions and not hybrid clones selected for by loss of dominant-acting growth suppressor genes. This finding was supported by detailed comparative genomic hybridization (CGH) studies, Q-banding karyotype analysis, and autofusions of representative clones. The purposeful creation of inherently unstable human-murine fusions between C8161 and B16-F1 where loss of putative

suppressor loci would be expected, resulted in fusions exhibiting decreased growth and non-metastatic behavior with progressive chromosomal loss. Neither p53, nm23, DNA methyltransferase, activated ras, fibroblast growth factor-4 (FGF-4), or epidermal growth factor receptor (EGFR) mediated the acquisition of the metastatic or undifferentiated phenotype within the C8161-human fusions. These studies are the first studies ever to successfully transfer the complete metastatic phenotype by somatic cell fusion and support the presence of a new high level regulatory pathway(s) involving dominant trans-acting factors which act pleiotropically to regulate an undifferentiated and highly metastatic phenotype.

L18 ANSWER 8 OF 24 MEDLINE

ACCESSION NUMBER: 97218145 MEDLINE
DOCUMENT NUMBER: 97218145 PubMed ID: 9065806
TITLE: Rat and human **maspins**: structures, **metastatic** suppressor activity and mutation in prostate cancer cells.
AUTHOR: Umekita Y; Hiipakka R A; Liao S
CORPORATE SOURCE: The Ben May Institute for Cancer Research , Department of Biochemistry and Molecular Biology, University of Chicago, IL 60637, USA.
CONTRACT NUMBER: CA58073 (NCI)
SOURCE: CANCER LETTERS, **(1997 Feb 26)** 113 (1-2) 87-93.
Journal code: 7600053. ISSN: 0304-3835.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U58857
ENTRY MONTH: 199704
ENTRY DATE: Entered STN: 19970422
Last Updated on STN: 19970422
Entered Medline: 19970407

AB The rat homologue of human **maspin** cDNA was cloned. The deduced amino acid sequence of rat **maspin** was homologous to human **maspin** with 88% of the amino acids conserved. Rat **maspin** mRNA was detected in rat mammary gland, vagina, urinary bladder, thymus, small intestine, skin, ventral prostate, seminal vesicles, and thyroid but not in many other organs, such as heart, lung, liver, brain and kidney. Rat **maspin** cDNA retrovirally introduced into highly metastatic Dunning AT3.1 rat prostate cancer cells did not suppress metastasis of these tumor cells in Copenhagen rats. **Maspin** mRNA was detected in 5/10 human prostatic carcinoma tissue samples. Two human prostate cancer cell lines, PC-3 and LNCaP, and two human prostatic carcinoma and two benign prostatic hyperplasia tissue samples contained **maspin** mRNA having an isoleucine to valine mutation at amino acid 319.

L18 ANSWER 12 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1997:56907 BIOSIS
DOCUMENT NUMBER: PREV199799356110
TITLE: Expression of **maspin** by human breast tumor cells inhibits primary tumor cell growth and lung **metastasis** in an athymic mouse model.
AUTHOR(S): Ding, I.; Huang, H. D.; Sabet, H.; Zou, Z. Q.; Zhang, L. R.; Kern, F. G.; Okunieff, P.
CORPORATE SOURCE: Radiation Oncol. Branch, NCI, NIH, Bethesda, MD USA
SOURCE: Blood, (1996) Vol. 88, No. 10 SUPPL. 1 PART 1-2, pp. 297B.
Meeting Info.: Thirty-eighth Annual Meeting of the American
Society of Hematology Orlando, Florida, USA December 6-10, 1996
ISSN: 0006-4971.
DOCUMENT TYPE: Conference; Abstract
LANGUAGE: English

L18 ANSWER 13 OF 24 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1996:254732 BIOSIS
DOCUMENT NUMBER: PREV199698810861
TITLE: Differential expression of **Maspin** protein in human adenocarcinomas and **squamous** cell carcinomas.
AUTHOR(S): Ding, I.; Zou, Z. Q.; Huang, H. D.; Zhang, K.; Zhang, L. R.; Tang, D.; Okunieff, P.
CORPORATE SOURCE: Natl. Cancer Inst., NIH, Bethesda, MD 20892 USA
SOURCE: Proceedings of the American Association for Cancer Research
Annual Meeting, (1996) Vol. 37, No. 0, pp. 90.
Meeting Info.: 87th Annual Meeting of the American Association for Cancer Research Washington, D.C., USA April
20-24, 1996
ISSN: 0197-016X.
DOCUMENT TYPE: Conference
LANGUAGE: English

L18 ANSWER 16 OF 24 SCISEARCH COPYRIGHT 2003 THOMSON ISI
ACCESSION NUMBER: 1998:80377 SCISEARCH
THE GENUINE ARTICLE: BK17U
TITLE: **Maspin** - A tumor suppressing serpin (Reprinted from Attempts to Understand **Metastasis** Formation I: **Metastasis**-Related Molecules, 1996)
AUTHOR: Sager R (Reprint); Sheng S; Pemberton P; Hendrix M J C
CORPORATE SOURCE: DANA FARBER CANC INST, DIV CANC GENET, 44 BINNEY ST, BOSTON, MA 02115 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: ADVANCES IN EXPERIMENTAL MEDICINE AND BIOLOGY, (**JAN 1997**) Vol. 425, pp. 77-88.
Publisher: PLENUM PRESS DIV PLENUM PUBLISHING CORP, 233 SPRING ST, NEW YORK, NY 10013.
ISSN: 0065-2598.
DOCUMENT TYPE: Reprint; Journal
LANGUAGE: English
REFERENCE COUNT: 15

L18 ANSWER 17 OF 24 SCISEARCH COPYRIGHT 2003 THOMSON ISI
ACCESSION NUMBER: 97:190621 SCISEARCH
THE GENUINE ARTICLE: WK895
TITLE: The role of serpin superfamily members in cancer
AUTHOR: Pemberton P A (Reprint)
CORPORATE SOURCE: LXR BIOTECHNOL, 1401 MARINA WAY S, RICHMOND, CA 94804 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: CANCER JOURNAL, (**JAN-FEB 1997**) Vol. 10, No. 1, pp. 24-30.
Publisher: ASSOC DEVELOPPEMENT COMMUNICATION CANCEROLOGIQUE, CANCER JOURNAL, 7 RUE GUY MOQUET, BP 8, 94801 VILLEJUIF, FRANCE.
ISSN: 0765-7846.
DOCUMENT TYPE: General Review; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 48

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The ''serpin'' class of serine proteinase inhibitors was originally shown to be involved in suppression of tumor invasion by their direct inhibitory actions on the matrix-degrading serine proteinases uPA and plasmin, It is now clear that individual members play a variety of roles in tumorigenesis, from regulating differentiation events, to inhibiting cysteine proteinases involved in matrix degradation and selection processes associated with tumor survival (apoptosis), Not all of these roles involve proteinase inhibition mediated by the serpin reactive site loop (RSL), instead other domains have been identified that confer these activities, and in some cases the RSL itself appears to have lost inhibitory activity and evolved a ligand-binding function, Several serpins involved in these processes map to the same locus on chromosome 18q21.3 (**maspin**, SCCA1, SCCA2, PAI-2), indicating that they arose by duplication of a common ancestral gene, It appears that these serpins and their target proteinases and/or ligands have evolved to control cellular proliferation and migration events and should therefore be examined more closely during tumor progression, and considered as key targets for the development of novel therapeutic anti-cancer agents.

L18 ANSWER 20 OF 24 CANCERLIT
ACCESSION NUMBER: 96653574 CANCERLIT
DOCUMENT NUMBER: 96653574
TITLE: Differential expression of **maspin** protein in human adenocarcinomas and **squamous** cell carcinomas (Meeting abstract).
AUTHOR: Ding I; Zou Z Q; Huang K D; Zhang K; Zhang L R; Tang D; Okunieff P
CORPORATE SOURCE: National Cancer Institute, Bethesda, MD 20892.
SOURCE: Proc Annu Meet Am Assoc Cancer Res, **(1996)** 37 A627.
ISSN: 0197-016X.
DOCUMENT TYPE: (MEETING ABSTRACTS)
LANGUAGE: English
FILE SEGMENT: Institute for Cell and Developmental Biology
ENTRY MONTH: 199609
ENTRY DATE: Entered STN: 19970509
Last Updated on STN: 19970509

AB Using the differential display method, **Maspin**, a member of the Serpin family of proteinase inhibitors, has been identified by screening the mRNA expression of normal human breast epithelium and tumor cells. Normal mammary epithelial cells, but not tumor cells or breast-derived fibroblasts, express **Maspin**. This distribution is consistent with the proposed tumor suppression associated with **Maspin** protein. In order to determine if the **Maspin** protein expression is important in carcinogenesis at other sites, 10 adenocarcinoma and 11 **squamous** cell carcinomas cell lines were tested for the **Maspin** protein expression by Western analysis. Adenocarcinoma cell lines included 7 ovarian and three endometrial tumor lines, and **squamous** cell tumors included 8 cervical carcinoma and 3 oral or skin carcinoma lines. None of the ovarian tumor lines (0/7) and only one of three of endometrial tumor lines had even moderate **Maspin** protein expression. However, seven of eight cervical carcinoma cell lines and two of three **squamous** cell carcinoma cell lines (SCC4 and A431) highly expressed the **Maspin** protein. Immunohistochemistry was done on tumor specimens from 27 ovarian and 9 esophageal carcinomas. 44% (12/27) of ovarian tumors and 77% (7/9) esophageal carcinoma specimens had either focal or diffuses **Maspin** immunoreactivity determined by an immunohistochemical staining using a polyclonal antibody. Our results indicate that the expression of **Maspin** plays a role in several human tumors, including ovary, cervix, endometrium, esophagus and tongue. The **Maspin** protein expression was less common in adenocarcinomas but was commonly overexpressed in **squamous** cell carcinomas.

L18 ANSWER 23 OF 24 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:654663 CAPLUS
DOCUMENT NUMBER: 132:120643
TITLE: New genes potentially involved in breast cancer **metastasis**
AUTHOR(S): Schwirzke, Marina; Schiemann, Sabine; Gnirke, Andrea U.; Weidle, Ulrich H.
CORPORATE SOURCE: Roche Pharmaceuticals, Penzberg, D-82372, Germany
SOURCE: Anticancer Research (**1999**), 19(3A), 1801-1814
CODEN: ANTRD4; ISSN: 0250-7005
PUBLISHER: International Institute of Anticancer Research
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review, with 135 refs. Identification of new genes involved in the pathogenesis of breast cancer opens new avenues for improved diagnostic markers and new mol. targets for improved treatment of this malignancy. In the following we review genes with proved involvement in invasion and metastasis of breast cancer as well as genes which exhibit an expression pattern that correlates with invasion and metastasis.
REFERENCE COUNT: 135 THERE ARE 135 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 24 OF 24 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:671816 CAPLUS
DOCUMENT NUMBER: 130:33609
TITLE: Identification of superior markers for polymerase chain reaction detection of breast cancer **metastases** in sentinel lymph nodes
AUTHOR(S): Min, C. Justus; Tafra, Lorraine; Verbanac, Kathryn M.
CORPORATE SOURCE: Departments of Biology, East Carolina University, Greenville, NC, 27858, USA
SOURCE: Cancer Research (**1998**), 58(20), 4581-4584
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Sentinel lymph node biopsy (SLNB) is being evaluated in breast cancer patients to improve detection of metastases and to guide therapy with minimal morbidity. The use of reverse transcription-PCR anal. to increase detection of tumor cells in SLN of breast cancer patients is hampered by the lack of specific markers. In this study, seven markers were evaluated by reverse transcription-PCR for expression in human breast adenocarcinoma lines (BrCa) and in normal nodes from non-cancer patients. Two markers yielded exceptional results; mammaglobin and carcinoembryonic antigen transcripts were detected in 100 and 71% BrCa, resp., and were absent from all normal lymph nodes. These markers will be used as components of a multimarker panel to evaluate sentinel nodes in an on-going, multicenter clin. trial.
REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 50 OF 64 MEDLINE
ACCESSION NUMBER: 90024082 MEDLINE
DOCUMENT NUMBER: 90024082 PubMed ID: 2802033
TITLE: Prognostic factors in squamous cell **carcinoma** of the larynx.
AUTHOR: Eiband J D; Elias E G; Suter C M; Gray W C; Didolkar M S
CORPORATE SOURCE: Department of Surgery, University of Maryland, Baltimore.
SOURCE: AMERICAN JOURNAL OF SURGERY, **(1989 Oct)** 158 (4) 314-7.
Journal code: 0370473. ISSN: 0002-9610.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198910
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19900328
Entered Medline: 19891031

AB One hundred fifty-two patients with squamous cell **carcinoma** of the larynx were studied. The disease-free survival and overall survival rates were correlated to 12 variables. Seven of them seemed to affect survival. Poor prognosis was related to (1) advanced stage of disease at diagnosis, (2) cord fixation and massive local invasion, (3) ulceration of the primary tumor, (4) **lymph node metastases** at diagnosis, (5) glottic lesions had a poorer prognosis than supraglottic ones, (6) locoregional recurrences, and (7) male gender. However, most of these significant differences were in disease-free **survival**, and only primary **tumor staging; lymph node** status; and locoregional recurrences affected overall survival. On the other hand, the other five variables showed no effect on either disease-free or overall survival rates. These included age, race, cell differentiation, type of recurrence, and the initial definitive therapeutic modality.

L15 ANSWER 52 OF 64 MEDLINE
ACCESSION NUMBER: 88159836 MEDLINE
DOCUMENT NUMBER: 88159836 PubMed ID: 2450417
TITLE: Is there a place for liver grafting for malignancy?.
AUTHOR: Pichlmayr R
CORPORATE SOURCE: Klinik fur Abdominal-und Transplantationschirurgie, Medizinische Hochschule, Hannover, FRG.
SOURCE: TRANSPLANTATION PROCEEDINGS, **(1988 Feb)** 20 (1 Suppl 1) 478-82.
Journal code: 0243532. ISSN: 0041-1345.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198804
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880411

AB In 94 patients liver transplantations for malignant tumors of the liver have been performed in this institution from 1972 to 1987. The long-term overall results in hepatic transplantation for irresectable tumors are disappointing in spite of good short-term palliation in most of the patients. Tumor recurrence is the rule. But individual long-living patients demonstrate the potentials of this treatment. Thus the crucial question will be a proper selection of patients. The relative suitability (in descending order of favorableness) of the kinds of tumors may range from HCC without cirrhosis, to central bile duct tumors, to HCC in cirrhosis, to CCC, and finally to secondaries. But this range can only give some probability for the success rate. More important is the **tumor stage**. **Survival** in **lymph node**-positive stages is by far worse than in **lymph node**-negative stages. The 6-month, 1-year, and 2-year actuarial survival data in our experience for **lymph node** -negative (**lymph node**-positive) HCC without cirrhosis are 83%, 75%, 75% (33%, 11%, 11%); in bile duct **carcinomas** in **lymph node**-negative stages (**lymph node** -positive) they are 6 months, 100% (40%); 1 year, 100% (13%); and 2 years, 83% (0%). Hepatic transplantation for selected tumor patients furthermore seems justified and is essential for a detailed analysis of the chance of different tumor types for success with this method of treatment.

L15 ANSWER 53 OF 64 MEDLINE

L15 ANSWER 53 OF 64 MEDLINE
ACCESSION NUMBER: 88251178 MEDLINE
DOCUMENT NUMBER: 88251178 PubMed ID: 3289517
TITLE: Surgical management of lung cancer with solitary cerebral **metastasis**.
AUTHOR: Hankins J R; Miller J E; Salcman M; Ferraro F; Green D C; Attar S; McLaughlin J S
CORPORATE SOURCE: Department of Surgery, University of Maryland School of Medicine, Baltimore.
SOURCE: ANNALS OF THORACIC SURGERY, **(1988 Jul)** 46 (1) 24-8. Ref: 20
Journal code: 15030100R. ISSN: 0003-4975.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198807
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880726

AB Between 1964 and 1986, 19 patients underwent resection of both a primary lung cancer and the associated brain **metastasis**. One patient underwent resection of 2 separate primary lung cancers and the associated **metastases**. The 12 men and 7 women ranged in age from 42 to 67 years (mean, 54.6 years). The cell type was adenocarcinoma in 12 tumors, squamous or adenosquamous cell in 5, large cell undifferentiated or anaplastic in 2, and malignant carcinoid in 1 tumor. The types of resection were as follows: lobectomy for 12 neoplasms, pneumonectomy for 5, bilobectomy for 2, and wedge resection for 1 neoplasm. Radiotherapy to the brain was given in connection with sixteen of the twenty craniotomies.

The patient with 2 separate primary neoplasms survived 19 years before dying 5 months after the second craniotomy. The mean survival is 8.0 +/- 2.1 years (+/- the standard error), and the median survival is 1.67 years.

Survival at 1 year was 65 +/- 10.7% and at 5 years, 45 +/- 11.1%. On univariate analysis, the following factors were found to correlate significantly with longer **survival**: a lung **tumor** in **Stage** I or II; negative mediastinal nodes; curative rather than palliative resection of the lung tumor; and age younger than 55 years. However, on multivariate analysis, only curative resection was a significant factor (p less than 0.01). We believe these results justify continued application of this combined surgical approach to patients having limited-stage lung cancer with a solitary brain **metastasis**.

L15 ANSWER 54 OF 64 MEDLINE
ACCESSION NUMBER: 89015968 MEDLINE
DOCUMENT NUMBER: 89015968 PubMed ID: 3140177
TITLE: Squamous cell **carcinoma** of the soft palate, uvula, and anterior faucial pillar.
AUTHOR: Weber R S; Peters L J; Wolf P; Guillamondegui O
CORPORATE SOURCE: Department of Head and Neck Surgery, University of Texas M.D. Anderson Hospital and Tumor Institute, Houston 77030.
SOURCE: OTOLARYNGOLOGY - HEAD AND NECK SURGERY, **(1988 Jul)** 99 (1) 16-23.
Journal code: 8508176. ISSN: 0194-5998.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198810
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19881031

AB This retrospective study concerns 188 patients with squamous cell **carcinoma** of the soft palate, uvula, and anterior faucial pillar treated for cure between 1970 and 1983. Men predominated in the group (1.9:1) and 55% of the patients were between 60 and 70 years old. Mean duration of followup was 56.7 months. TNM stage distribution was 29, 67, 37, and 49 patients for stages I, II, III, and IV respectively; six patients were unstaged because of previous excisional biopsy. Treatment to the primary site consisted of radiotherapy for 150 patients, surgery alone for 28 patients, and combined therapy for 10 patients. Primary control for T stages 1 through 4 was: 91% (31 of 34), 77% (71 of 92), 77% (30 of 39), and 35% (6 of 17), respectively. One hundred twenty-eight patients were N0 at presentation, as compared to 60 patients with regional nodal **metastasis**. Regional control was obtained in 87.5% of patients with N0 necks and in 76.7% of those with nodal involvement. In patients with primary control, these figures were 89% and 81%. Overall determinant survival was 80% at 2 years, but fell to 67% at 5 years. In addition to advanced **tumor stage**, the **survival** rate was reduced by regional **lymph node metastasis**. Tumor extension to the tongue base diminished survival. Survival was poorer among patients with midline tumors or tumors that extended across the palatine arch (37 patients) than for those with unilateral primary tumors (151 patients) (p less than 0.05). Despite similar T-stage distribution, the incidence of regional nodal **metastasis** was 49% in the former group, compared with 28% in the latter.(ABSTRACT TRUNCATED AT 250 WORDS)

L15 ANSWER 55 OF 64 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1988:73232 BIOSIS
DOCUMENT NUMBER: BA85:39531
TITLE: **CARCINOMA** OF THE COLON LONG-TERM SURVIVAL AND PROGNOSIS AFTER SURGICAL TREATMENT IN A SERIES OF 798 PATIENTS.
AUTHOR(S): MOREAUX J; CATALA M
CORPORATE SOURCE: CENT. MEDICO-CHIRURGICAL DE LA PORTE DE CHOISY, 15 AVENUE DE LA PORTE DE CHOISY, 75013 PARIS, FR.
SOURCE: WORLD J SURG, (1987) 11 (6), 804-808.
CODEN: WJSUDI. ISSN: 0364-2313.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB From 1964 to 1985, a total of 798 patients (405 female, 393 male) were operated on for a single cancer of the colon. Fifty-eight percent of the patients were between 60 and 80 years of age. Liver and/or peritoneal **metastases** were present in 16.3% of the 818 cases. Resection was performed in 754 cases (92.2%), and was considered to be curative in 646 (78.9%). Tumors were differentiated in 90.5% of the cases. Regional **lymph nodes** were involved in 33.3% and serosal penetration was present in 19.5% of the cases. There were 7 postoperative deaths, 3 (0.5%) of them after curative resection. The actuarial curves of survival showed a probability of survival after all operations of 62% at 5 years and 46% at 10 years, and after curative resection of 78% at 5 years and 58% at 10 years. Prognosis has been established from the 513 patients operated on before 1980; follow-up data were available for all but 4 of them. Tumor site in the right or left colon did not relate significantly to **survival**. **Tumor staging** was the main prognostic factor. The 5-year survival rate was 40% in patients with positive nodes, 74.7% in those with negative nodes ($p < 0.001$), 97.6% in those with invasion limited to mucosa or submucosa, and 41.9% in those with serosal invasion ($p < 0.001$). Based on Dukes' classification, the 5-year survival rates for A, B, C, and D tumors were 91%, 76.7% ($p = 0.01$), 53.1% ($p < 0.001$), and 4.7% ($p < 0.001$), respectively. Time elapsed between first symptom and operation did not relate significantly to survival. Prognosis was better in patients less than 50 years old when compared with patients 50-70 years of age ($p < 0.01$), and was better in female patients than in male patients ($p = 0.02$).

L15 ANSWER 49 OF 64 MEDLINE DUPLICATE 23

ACCESSION NUMBER: 90068906 MEDLINE
DOCUMENT NUMBER: 90068906 PubMed ID: 2587716
TITLE: [Percutaneous radiotherapy for thyroid gland **carcinoma**].
Ergebnisse der perkutanen Strahlentherapie bei Schilddrusenkarzinomen.
AUTHOR: Kleinert G
SOURCE: RADIOBIOLOGIA, RADIOTHERAPIA, **(1989)** 30 (5) 473-80.
Journal code: 0401247. ISSN: 0033-8184.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199001
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19900328
Entered Medline: 19900110

AB Prognostically relevant factors as well as indications for percutaneous radiotherapy are analysed by the hand of a retrospective analysis of therapeutic results in 86 patients that were exposed a percutaneous radiotherapy because of a thyroid **carcinoma** at the Clinic and Policlinic of the Medical Academy Erfurt during the period 1972 to 1982. The 5-years-survivals of 83% for patients with differentiated **carcinoma** and 22% for patients with dedifferentiated **carcinoma** prove the influence of tumor histology on prognosis of the disease. Next to it the locoregional tumor spreading at beginning of therapy rendered prognostically relevant. The 5-years-**survival** was 83% in **tumor stages** T1-3N0M0. With **metastatic** infiltration into **lymph-nodes** of the neck the 5-years-survival decreased to 57%, with spreading of the primary tumor beyond organ borders to 23.5%. The postoperative percutaneous radiotherapy should be applied in all cases of **metastatic** infiltration of **lymph-nodes**. In large, inoperable tumors the percutaneous radiotherapy is the solely possible palliative measure that should be applied both in differentiated and also in anaplastic **carcinomas** in spite of infaust prognosis.

L15 ANSWER 59 OF 64 MEDLINE
ACCESSION NUMBER: 85095913 MEDLINE
DOCUMENT NUMBER: 85095913 PubMed ID: 2981516
TITLE: Surgical therapeutic planning for non-small cell lung cancer.
AUTHOR: Sawamura K; Mori T; Hashimoto S; Iuchi K; Tada H; Lee Y E; Mizuta T; Ichimiya A; Akashi A
SOURCE: GAN TO KAGAKU RYOHO [JAPANESE JOURNAL OF CANCER AND CHEMOTHERAPY], **(1985 Jan)** 12 (1) 36-44.
Journal code: 7810034. ISSN: 0385-0684.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Japanese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198502
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19850214

AB The survival rates of 380 resected cases of lung cancer in our hospital were analyzed according to curability and histological cell type. The overall 5-year survival rate for stage I a cases was 64.5%, that for stage I b 52.3%, and that for stage II 26.7%. However, there were distinct differences in survival rates between stages I a-II with mediastinal **lymph node** dissection and those without mediastinal **lymph node** dissection. Of these 380 tumors, many were advanced (for instance, stage III tumors comprised 180 cases). T3 tumors had better prognosis (40.7% showing 5-year survival) than N2 tumors (26.7% showing 5-year **survival**). Among **stage** III **tumors**, squamous cell **carcinoma** (T3: 41%, N2: 36.7% showing 5-year survival) had a better prognosis than adenocarcinoma (T3: 16.1%, N2: 21.4%). T3N2 tumors, however, had such a poor prognosis that the value of surgery in these cases seemed questionable. Adjuvant therapy should therefore be evaluated accurately in future to improve prognosis. It was stressed that a randomized controlled study would be needed to evaluate the effectiveness of adjuvant therapy.

L15 ANSWER 61 OF 64 MEDLINE DUPLICATE 24
ACCESSION NUMBER: 82276071 MEDLINE
DOCUMENT NUMBER: 82276071 PubMed ID: 6810406
TITLE: Advanced **carcinoma** of the nasopharynx. A clinical study of 274 patients.
AUTHOR: Petrovich Z; Cox J D; Roswit B; MacKintosh R; Middleton R; Ohanian M; Rao Y; Byhardt R W; Paig C; del Regato J A
SOURCE: RADIOLOGY, **(1982 Sep)** 144 (4) 905-8.
Journal code: 0401260. ISSN: 0033-8419.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198210
ENTRY DATE: Entered STN: 19900317
Last Updated on STN: 19900317
Entered Medline: 19821012

AB A total of 274 patients with a diagnosis of nasopharyngeal **carcinoma** was treated in eight Veterans Administration Hospitals over a period of 22 years. Of the 274 patients, 256 (93%) had squamous-cell **carcinoma**, while 18 (7%) had other tumors. Most of the squamous-cell **carcinoma** patients (82%) had Stage IV disease; cervical **lymph node metastases** were found in 193 (75%), and distant **metastases** were present in 22 (9%). The actuarial 5-, 10-, and 15-year survival rates for the 256 squamous-cell **carcinoma** patients were 15%, 10%, and 7%, while they were 49%, 42%, and 35% for the 18 patients with other tumors (p = 0.006). There was a progressive decrease in 5-year survival with the increase in the **stage** of **tumor**. The **survival** of the 63 patients without **metastases** was better than the survival of the 193 patients with cervical **metastases** (24% vs. 12% at 5 years, p = 0.03). The presence of T4 disease or Initial Performance Status of less than 80 on the Karnofsky Scale indicated a poor prognosis (p = 0.0001). Treatment failure occurred in 83% of the patients by 2 years after therapy and was due to the lack of tumor control at the primary site. Advanced (N3) cervical **lymph node metastases** indicated that systemic tumor dissemination of the nasopharynx is an uncommon malignancy.

L4 ANSWER 2 OF 6 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2000:270286 BIOSIS
DOCUMENT NUMBER: PREV200000270286
TITLE: High tumor **maspin** expression is associated with improved survival of patients with **oral** **squamous** cell carcinoma.
AUTHOR(S): Xia, Weiya (1); Lau, Y.-K. (1); Hu, M. C.-T. (1); Li, L. (1); Johnston, D. A. (1); Sheng, S.-J. (1); El-Naggar, A. K. (1); Hung, M. C. (1)
CORPORATE SOURCE: (1) M D Anderson Cancer Ctr, Univ of Texas, Houston, TX USA
SOURCE: Proceedings of the American Association for Cancer Research
Annual Meeting, (March, 2000) No. 41, pp. 689. print..
Meeting Info.: 91st Annual Meeting of the American Association for Cancer Research. San Francisco, California,
USA April 01-05, 2000
ISSN: 0197-016X.
DOCUMENT TYPE: Conference
LANGUAGE: English
S

L4 ANSWER 4 OF 6 MEDLINE DUPLICATE 3

ACCESSION NUMBER: 1999458661 MEDLINE
DOCUMENT NUMBER: 99458661 PubMed ID: 10527881
TITLE: Identification and cDNA cloning of headpin, a novel differentially expressed serpin that maps to chromosome 18q.
AUTHOR: Spring P; Nakashima T; Frederick M; Henderson Y; Clayman G
CORPORATE SOURCE: Department of Head and Neck Surgery, M. D. Anderson Cancer Center, Houston, Texas, 77030, USA.
CONTRACT NUMBER: 1P50DE11906-01 (NIDCR)
CA16672 (NCI)
R29DE11689-01A1 (NIDCR)
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1999 Oct 14) 264 (1) 299-304.
Journal code: 9Y8; 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF169949
ENTRY MONTH: 199911
ENTRY DATE: Entered STN: 20000111
Last Updated on STN: 20000111
Entered Medline: 19991112

AB Differential display was used to identify a novel serpin (headpin) underexpressed in **squamous** cell cancers of the **oral** cavity. Headpin cDNA encoding a complete open reading frame was cloned and sequenced. Headpin is expressed in normal **oral** mucosal tissue, skin, and cultured keratinocytes. Using Northern analysis and relative reverse-transcription polymerase chain reaction (relative RT-PCR), downregulation of headpin mRNA expression was demonstrated in **oral** cavity **squamous** carcinomas. Northern blot analysis identified a 3. 3-kb headpin mRNA transcript. Headpin is a 391-amino-acid protein with a theoretical molecular weight of 44 kDa. Hinge region homology at the reactive site loop suggests that headpin belongs to the inhibitory class of serine protease inhibitors. Headpin was mapped to 18q21.3/18q22. This region includes the ovalbumin serpins (ov-serpins) **maspin**, SCCA1, SCCA2, and PAI-2. Furthermore, 18q is recognized as a region for frequent loss of heterozygosity (LOH) in **head** and **neck** cancer and other malignancies.

L4 ANSWER 6 OF 6 CANCERLIT
ACCESSION NUMBER: 96653574 CANCERLIT
DOCUMENT NUMBER: 96653574
TITLE: Differential expression of **maspin** protein in human adenocarcinomas and squamous cell carcinomas (Meeting abstract).
AUTHOR: Ding I; Zou Z Q; Huang K D; Zhang K; Zhang L R; Tang D; Okunieff P
CORPORATE SOURCE: National Cancer Institute, Bethesda, MD 20892.
SOURCE: Proc Annu Meet Am Assoc Cancer Res, (1996). Vol. 37, pp. A627.
ISSN: 0197-016X.
DOCUMENT TYPE: (MEETING ABSTRACTS)
FILE SEGMENT: ICDB
LANGUAGE: English
ENTRY MONTH: 199609

AB Using the differential display method, **Maspin**, a member of the Serpin family of proteinase inhibitors, has been identified by screening the mRNA expression of normal human breast epithelium and tumor cells. Normal mammary epithelial cells, but not tumor cells or breast-derived fibroblasts, express **Maspin**. This distribution is consistent with the proposed tumor suppression associated with **Maspin** protein. In order to determine if the **Maspin** protein expression is important in carcinogenesis at other sites, 10 adenocarcinoma and 11 **squamous** cell carcinomas cell lines were tested for the **Maspin** protein expression by Western analysis. Adenocarcinoma cell lines included 7 ovarian and three endometrial tumor lines, and **squamous** cell tumors included 8 cervical carcinoma and 3 **oral** or skin carcinoma lines. None of the ovarian tumor lines (0/7) and only one of three of endometrial tumor lines had even moderate **Maspin** protein expression. However, seven of eight cervical carcinoma cell lines and two of three **squamous** cell carcinoma cell lines (SCC4 and A431) highly expressed the **Maspin** protein. Immunohistochemistry was done on tumor specimens from 27 ovarian and 9 esophageal carcinomas. 44% (12/27) of ovarian tumors and 77% (7/9) esophageal carcinoma specimens had either focal or diffuses **Maspin** immunoreactivity determined by an immunohistochemical staining using a polyclonal antibody. Our results indicate that the expression of **Maspin** plays a role in several human tumors, including ovary, cervix, endometrium, esophagus and tongue. The **Maspin** protein expression was less common in adenocarcinomas but was commonly overexpressed in **squamous** cell carcinomas.

L3 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:42639 BIOSIS
DOCUMENT NUMBER: PREV199800042639
TITLE: **Maspin** is an intracellular serpin that partitions into secretory vesicles and is present at the cell surface.
AUTHOR(S): Pemberton, Philip A. (1); Tipton, A. Rene; Pavloff, Nadine; Smith, Jason; Erickson, James R.; Mouchabeck, Zahi M.; Kiefer, Michael C.
CORPORATE SOURCE: (1) LXR Biotechnol. Inc., 1401 Marina Way S., Richmond, CA 94804 USA
SOURCE: Journal of Histochemistry and Cytochemistry, (**Dec., 1997**) Vol. **45**, No. 12, pp. **1697** -1706.
ISSN: 0022-1554.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The tumor suppressor **maspin** (mammary serpin) was originally identified as a component of human mammary epithelial cells that is downregulated as mammary tumor cells progress from the benign to the invasive and metastatic states. **Maspin** inhibits cellular invasion, motility, and proliferation, but its mechanism of action is currently unknown. Because the cellular machinery responsible for these processes is cytoplasmic, we have reexamined the tissue distribution and subcellular localization of **maspin**. We find that **maspin**, or a **maspin**-like protein, is present in many human organs, in which it localizes to epithelia. In cultured human mammary myoepithelial cells, **maspin** is predominantly a soluble cytoplasmic protein that associates with secretory vesicles and is present at the cell surface. In vitro assays show that the vesicle association is due to the existence of an uncleaved facultative secretion signal that allows small amounts of **maspin** to partition into the endoplasmic reticulum. These results demonstrate that **maspin** is more widespread than previously believed. The subcellular localization studies indicate that soluble intracellular and vesicle associated **maspin** probably play an important role in controlling the invasion, motility, and proliferation of cells expressing it, whereas extracellular **maspin** may also regulate these processes in adjacent cells.

L12 ANSWER 79 OF 79 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1978:247280 BIOSIS
DOCUMENT NUMBER: BA66:59777
TITLE: DIFFERENCES IN PATHOLOGICAL CHARACTERISTICS AND PROGNOSIS OF CLINICAL A-2 PROSTATIC CANCER FROM A-1 AND B DISEASE.
AUTHOR(S): GOLIMBU M; SCHINELLA R; MORALES P; KURUSU S
CORPORATE SOURCE: DEP. UROL., N.Y. UNIV. MED. CENT., N.Y. VETERANS ADM. HOSP., NEW YORK, N.Y., USA.
SOURCE: J UROL, (1978) 119 (5), 618-622.
CODEN: JOURAA. ISSN: 0022-5347.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB A retrospective study was done of 53 cases of clinical stages A1 to B2 prostatic carcinomas staged by pelvic lymphadenectomy. The study compared the histologic differentiation, degree of lymphocytic infiltration, incidence of **lymph node metastases** and type of cellular response of clinical stage A2 to stages A1 and B disease. The available data pertaining to the incidence and survival of patients with stage A2 prostatic carcinoma were analyzed. One of every 3 unsuspected carcinomas is of clinical stage A2. The stage A2 tumors are diffused, with a higher degree of undifferentiation and a higher incidence of **lymph node metastases** than **tumors** classified as **stage** A1 and B1. **Survival** of patients with clinical **stage** A2 **tumors** is lower than **survival** of patients with clinical stage B1 disease. Clinical stage A2 tumors are more advanced biologically than clinical stage B1 tumors.

L11 ANSWER 4 OF 23 PCTFULL COPYRIGHT 2003 Univentio
ACCESSION NUMBER: 1998011238 PCTFULL ED 20020514
TITLE (ENGLISH): PROTEASE M, A NOVEL SERINE PROTEASE
TITLE (FRENCH): PROTEASE M, UNE NOUVELLE SERINE PROTEASE
INVENTOR(S): ANISOWICZ, Anthony;
SAGER, Ruth;
SOTIROPOULOU, Georgia
PATENT ASSIGNEE(S): DANA-FARBER CANCER INSTITUTE
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

	NUMBER	KIND	DATE
	WO 9811238	A2	19980319

DESIGNATED STATES
W: AU CA JP AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
APPLICATION INFO.: WO 1997-US16175 A 19970911
PRIORITY INFO.: US 1996-60/025,301 19960913

ABEN Isolated nucleic acid molecules encoding a novel serine protease, Protease M, is disclosed.
Protease M is downregulated in metastatic mammary epithelial tumor cells, as well as other tumor
cells, and is upregulated in senescent cells. In addition to isolated nucleic acid molecules, the
invention provides antisense nucleic acid molecules, recombinant expression vectors containing a
nucleic acid molecule of the invention, host cells into which the expression vectors have been
introduced and non-human transgenic animals in which a Protease M gene has been introduced or
disrupted. The invention further provides isolated Protease M proteins, fusion proteins, antigenic
peptides and anti-Protease M antibodies. Diagnostic assays, drug screening assays, and therapeutic
methods utilizing compositions of the invention are also provided.
ABFR Molecules nucleotidiques isolees codant une nouvelle serine protease, la
protease M. Cette
protease est obtenue par regulation negative dans des cellules metastatiques de tumeurs epitheliales
mammaires, ainsi que dans d'autres cellules tumorales, et elle est obtenue par regulation positive
dans des cellules senescentes. On decrit par ailleurs des molecules nucleotidiques anti-sens, des
vecteurs d'expression recombines contenant une molecule nucleotidique visee dans l'invention, des
cellules hotes dans lesquelles les vecteurs d'expression ont ete introduits, et des animaux
transgeniques non humains dans lesquels on a introduit ou dissocie un gene de protease M. On decrit
par ailleurs des proteines de protease M isolees, des proteines fusionnees, des peptides
antigeniques et des anticorps anti-protease M, et par ailleurs, des dosages diagnostiques, des
methodes de criblage de medicaments ainsi que des procedes therapeutiques faisant appel aux

compositions specifiees.

DETD . . . DESCRIPTION Protease M RNA 6A2 RNA EXP
EXP
T24 bladder transitional cell - -
carcinoma
A549(CCL 1 85) lung carcinoma - -
Calu- I lung **epidermoid carcinoma** - -
Oat 4 lung small cell carcinoma - -
G-361 malignant melanoma - -
SMKE 30 malignant melanoma - -
A2058 malignant melanoma - -
SCC-25 tongue **squamous** cell - -
carcinoma
RD rhadomyosarcoma of pelvis - -
Kaposi kaposis sarcoma - -
FS3 foreskin fibroblast - -
Leukocyte normal leukocytes - -
- 72 -
TABLE 5. SHOWS RNA EXPRESSION IN MAMMARY TISSUE
SAMPLE TYPE Protease **MASPIN** CX26 CX43
M
8IN N cell strain ++ +++-+- +++ ....

L11 ANSWER 7 OF 23 PCTFULL COPYRIGHT 2003 Univentio
ACCESSION NUMBER: 1995020041 PCTFULL ED 20020514
TITLE (ENGLISH): IMMORTALIZED HUMAN MYOEPITHELIAL CELLS AND THEIR USES
TITLE (FRENCH): CELLULES MIOEPITHELIALES HUMAINES IMMORTALISEES
INVENTOR(S): BARSKY, Sanford, H.;
STERNLICHT, Mark
PATENT ASSIGNEE(S): THE REGENTS OF THE UNIVERSITY OF CALIFORNIA
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

	NUMBER	KIND	DATE
	WO 9520041	A1	19950727

DESIGNATED STATES
W: JP AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE
APPLICATION INFO.: WO 1995-US858 A 19950120
PRIORITY INFO.: US 1994-8/184,720 19940121

ABEN Methods and compositions are provided for the culture of human primary carcinomas and in situ carcinomas. Feeder layers derived from a human parotid basal cell carcinoma, having the HMS-1 phenotype, are able to support the growth of the primary carcinomas, and allow for spheroid formation. Invasion inhibiting factors active against human tumors, derived from HMS-1, are also provided. Human basement membrane and extracellular matrix is provided, produced by a tumorigenic cell line, where the basement membrane and extracellular matrix can be used for the growth of a variety of cells, in culture and in vivo. Other related cell lines are provided, which can serve to evaluate in vivo the response of tumorigenic cells to various agents, including basement membrane and extracellular matrix. The basement membrane and extracellular matrix finds use in allowing the growth of cells in culture and in vivo, particularly cells which are otherwise refractory to xenografting.

ABFR L'invention concerne des procedes et des compositions permettant la culture de carcinomes primaires humains et de carcinomes in situ. Des couches nourricieres derivees d'un carcinome basocellulaires de parotyde humaine, presentant le phenotype HMS-1, peuvent soutenir la croissance des carcinomes primaires, et permettent la formation de spheroides. Des facteurs inhibant l'invasion, agissant contre les tumeurs humaines, derives de HMS-1, sont egalement decrits. L'invention concerne egalement une membrane basale et une matrice extracellulaire humaine, produite par une lignee cellulaire tumorigene, cette membrane basale et cette matrice extracellulaire pouvant etre utilisees pour permettre la croissance d'une variete de cellules, en culture et in vivo.

L'invention concerne egalement d'autres lignees cellulaires qui peuvent servir pour evaluer in vivo
la reponse de cellules tumorigenes a divers agents, y compris ladite membrane basale et ladite
matrice extracellulaire. Cette membrane basale et cette matrice extracellulaire sont utilisees pour
permettre la croissance de cellules en culture et in vivo, en particulier de cellules qui autrement
sont refractaires a l'heterogreffe.

DETD **Maspin** mRNA expression was determined by northern blot analysis using a I
kb **maspin** cDNA probe. The expression of TIMP- 1 was also determined, using the
0.7 kb cDNA insert of pEPA. Poly-A selected rnRNA. . . gel, transferred to nylon membrane and hybridized with
32p-labeled probe. Protease inhibitor expression was examined for rnRNA isolated
from HMS-1, salivary gland **epidermoid carcinoma**
cells (A253; ATCC HTB 41),
normal human prostate derived fibroblasts (NHF), MDA-MB-231 breast adenocarcinorna cells (ATCC HTB 26), and a human diploid. . .

L14 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1997:202651 CAPLUS
DOCUMENT NUMBER: 126:221310
TITLE: mMaspin: The mouse homolog of a human tumor suppressor gene inhibits mammary tumor invasion and motility
AUTHOR(S): Zhang, Ming; Sheng, Shijie; Maass, Nicolai; Sager, Ruth
CORPORATE SOURCE: Dana Farber Cancer Institute, Harvard Medical School, Boston, MA, 02115, USA
SOURCE: Molecular Medicine (New York) (1997), 3(1), 49-59
CODEN: MOMEF3; ISSN: 1076-1551
PUBLISHER: Springer
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The human **maspin** gene encodes a protein in the serine proteinase inhibitor (serpin) family with tumor-suppressing functions in cell culture and in nude mice. In order to examine the role of **maspin** in an intact mammal, we cloned and sequenced the cDNA of mouse **maspin**. The recombinant protein was produced and its activity in cell culture was assessed. Mouse **maspin** (mMaspin) was cloned by screening a mouse mammary gland cDNA library with the human **maspin** cDNA probe. Northern blot anal. was used to examine the expression patterns in mouse tissues, mammary epithelial cells, and carcinomas. Recombinant mMaspin protein was produced in E. coli. Invasion and motility assays were used to assess the biol. function of mMaspin. MMaspin is 89% homologous with human **maspin** at the amino acid level. Like its human homolog, mMaspin is expressed in normal mouse mammary epithelial cells and down-regulated in mouse breast tumor cell lines. The expression is altered at different developmental stages in mammary gland. Addn. of the recombinant mMaspin protein to mouse tumor cells was shown to inhibit invasion in a dose-dependent manner. As with the human protein, recombinant mMaspin protein also inhibited mouse mammary tumor motility. Deletion in the putative mMaspin reactive site loop (RSL) region resulted in the loss of its inhibitory functions. MMaspin is the mouse homolog of a human tumor suppressor gene. The expression of mMaspin is down-regulated in tumor cells and is altered at different developmental stages of mammary gland. MMaspin has inhibitory properties similar to those of human **maspin** in cell culture, suggesting that the homologous proteins play similar physiol. roles in vivo.

Cancer Res. 1981 May;41(5):1657-63.

Related Articles,

Links

Tumorigenic keratinocyte lines requiring anchorage and fibroblast support cultures from human squamous cell carcinomas.

Rheinwald JG, Beckett MA.

We have established cell lines from six human squamous cell carcinomas (SCC) of the epidermis and tongue, using culture methods previously developed for clonal growth and serial cultivation of normal keratinocytes. The SCC lines all form rapidly growing, well-differentiated SCC's or progressively growing squamous cysts in nude mice. In contrast to normal keratinocytes, SCC cells form unstratified or very poorly stratifying colonies and do not require epidermal growth factor for sustained growth. The SCC lines vary in their requirement for a fibroblast feeder layer to support clonal growth, as normal keratinocytes possess. Only one line forms large, progressively growing colonies at high efficiency in semisolid medium; the other five lines exhibit only a small amount of abortive growth in semisolid medium, after which the cells appear to rapidly degenerate. These results demonstrate that SCC's often grow as established lines in culture, but they frequently possess in vitro growth requirements similar to those of normal keratinocytes. Consequently, neither semisolid medium nor standard surface culture media are appropriate for initiating primary SCC cultures or for selecting transformants out of carcinogen-treated keratinocyte populations, because they do not provide conditions permissive for the growth of many malignant keratinocytes.

MeSH Terms:
- Animal
- Carcinoma, Squamous Cell/pathology*
- Cell Adhesion
- Cell Division
- Cells, Cultured
- Epidermis/pathology
- Fibroblasts/pathology
- Head and Neck Neoplasms/pathology*
- Human
- Mice
- Mice, Nude
- Neoplasms, Experimental/pathology
- Skin Neoplasms/pathology*
- Support, Non-U.S. Gov't
- Support, U.S. Gov't, P.H.S.

Grant Support:
- CA-19589/CA/NCI
- CA-26656/CA/NCI

J Natl Cancer Inst. 1973 Nov;51(5):1417-23.

Related Articles,

Links

In vitro cultivation of human tumors: establishment of cell lines derived from a series of solid tumors.

Giard DJ, Aaronson SA, Todaro GJ, Arnstein P, Kersey JH, Dosik H, Parks WP.

MeSH Terms:
- Astrocytoma
- Brain Neoplasms
- Carcinoma
- Carcinoma, Squamous Cell
- Cell Division
- Cell Line*
- Cell Transformation, Neoplastic
- Cells, Cultured
- Chromosome Aberrations
- Epithelial Cells
- Fibroblasts
- Glioblastoma
- Human
- Immune Sera
- Immunosuppression
- Kidney Neoplasms
- Lung Neoplasms
- Melanoma
- Neoplasm Transplantation
- Neoplasms*
- Neoplasms, Experimental
- Rhabdomyosarcoma
- Sarcoma
- Skin/cytology
- T-Lymphocytes/immunology
- Transplantation, Heterologous

Substances:
- Immune Sera

PMID: 4357758 [PubMed - indexed for MEDLINE]

L14 ANSWER 4 OF 31 MEDLINE

ACCESSION NUMBER: 82202117 MEDLINE

DOCUMENT NUMBER: 82202117 PubMed ID: 6952723

TITLE: **Squamous carcinoma** of the **breast**: diagnosis by aspiration cytology.

AUTHOR: Leiman G

SOURCE: ACTA CYTOLOGICA, **(1982 Mar-Apr)** 26 (2) 201-9.
Journal code: 0370307. ISSN: 0001-5547.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 198207

ENTRY DATE: Entered STN: 19900317
Last Updated on STN: 19900317
Entered Medline: 19820708

AB Squamous carcinoma is a rarely encountered lesion in the breast, and its diagnosis by aspiration cytology is unreported. Six cases of **squamous carcinoma** occurring in the **breast**, all diagnosed preoperatively by cytology, are discussed. Cytologic and histopathologic features of both primary and secondary carcinomas, pure and metaplastic types, are described, together with aspects of etiology, morphology and prognosis.

14 ANSWER 8 OF 31 MEDLINE
ACCESSION NUMBER: 82025282 MEDLINE
DOCUMENT NUMBER: 82025282 PubMed ID: 7284964
TITLE: Primary **squamous** cell **carcinoma** of the **breast**.
AUTHOR: Toikkanen S
SOURCE: CANCER, **(1981 Oct 1)** 48 (7) 1629-32.
Journal code: 0374236. ISSN: 0008-543X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198112
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19811221

AB Upon reexamination of about 4000 **breast** cancer biopsies, three pure primary **squamous** cell **carcinomas** (SCC) were found. The light and electron microscopic findings of these three cases are described. The carcinomas seemed to originate from the glandular tissue of the breast and followed an extremely aggressive clinical course.

SCC must be regarded as a separate entity distinct from adenocarcinoma of the breast with squamous cell metaplasia.

L6 ANSWER 4 OF 5 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97202469 MEDLINE
DOCUMENT NUMBER: 97202469 PubMed ID: 9049988
TITLE: The myoepithelial defense: a host defense against cancer.
AUTHOR: Sternlicht M D; Barsky S H
CORPORATE SOURCE: Department of Pathology, UCLA School of Medicine 90024, USA.
SOURCE: MEDICAL HYPOTHESES, (1997 Jan) 48 (1) 37-46.
Journal code: 7505668. ISSN: 0306-9877.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199705
ENTRY DATE: Entered STN: 19970523
Last Updated on STN: 19970523
Entered Medline: 19970512

AB The behavior of human tumors depends not only on the nature of the tumor cells themselves but also on the modifying effects of various normal host cells such as fibroblasts and endothelial cells. One cell type, however--the myoepithelial cell--has not been studied scientifically. Myoepithelial cells normally surround ducts and acini of glandular organs such as the breast and salivary glands and contribute to the synthesis of a surrounding basement membrane. This relationship suggests that myoepithelial cells may exert paracrine effects on **glandular epithelium** and also regulate the progression of ductal carcinoma in situ (DCIS) to invasive carcinoma. Myoepithelial tumors, in turn, tend to be benign or low-grade neoplasms that exhibit the rare property of accumulating rather than degrading extracellular matrix material. To better understand the nature of myoepithelial tumors, as well as the possible role of normal myoepithelial host cells in cancer, we have established immortal cell lines and a number of transplantable xenografts from various human myoepithelial tumors of the salivary gland and breast. The cell lines exhibit a normal myoepithelial phenotype and the xenografts continue to accumulate an abundant extracellular matrix. Further ultrastructural, immunocytochemical, molecular, and biochemical studies reveal that myoepithelial cells secrete relatively low levels of matrix-degrading proteinases but relatively high levels of **maspin** and various other anti-invasive proteinase inhibitors, that some of these inhibitors accumulate within the myoepithelial matrix, and that myoepithelial cells can induce epithelial morphogenesis (spheroid formation) and inhibit tumor-cell invasion in vitro. Myoepithelial cells, which surround normal breast ducts and DCIS, have also been found to selectively express **maspin** and certain proteinase inhibitors in situ. These inherent myoepithelial properties are likely to contribute to the low-grade nature of myoepithelial neoplasms and advance our hypothesis that host myoepithelial cells regulate the progression of in situ to invasive carcinoma by providing an important host defense against cancer invasion.